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Intestinal and gastric tonometry during experimental burn shock☆

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ARTICLE INFO

Article history:
Accepted 26 November 2006

Keywords:
Burns
Small intestine
Gastric mucosa
Intestinal mucosa
Shock

ABSTRACT

Introduction: The occurrence of organ failure following thermal injury, despite restoration of hemodynamic parameters and urine output during resuscitation, has led to efforts to measure end-organ perfusion. The purpose of this 24-h study was to evaluate the utility of gastrointestinal (GI) tonometry during burn shock and resuscitation.

Methods: Male swine ($n = 11$, 23.3 ± 0.9 kg) were anesthetized with ketamine and propofol. A 70% full thickness burn was caused by immersion in 97 °C water for 30 s. Resuscitation with lactated Ringer's, 4 ml/kg/% burn, was begun at hour 6 and titrated to urine output (UO). Arterial blood gases and pulmonary artery catheter data were measured every 6 h. Gastric and ileal regional PCO_2 ($PrCO_2$) were measured continuously by air tonometry, and the gastric and ileal intramucosal pH (pHi) and PCO_2 gap ($PrCO_2 - PaCO_2$) were calculated every 6 h.

Results: Gastric pHi, ileal $PrCO_2$, ileal pHi, and ileal PCO_2 gap (but not gastric $PrCO_2$ or PCO_2 gap) all decreased with shock and were restored to baseline levels by resuscitation. Changes in ileal $PrCO_2$ were of greater magnitude and demonstrated decreased variability than those in gastric $PrCO_2$.

Conclusions: In this model, ileal tonometry outperformed gastric tonometry during burn shock and resuscitation.

1. Introduction

Despite fluid resuscitation adjusted to maintain an adequate urine output and hemodynamic stability, systemic metabolic acidosis and regional ischemia have been well documented during burn shock, and were associated with increased mortality, systemic inflammatory response syndrome (SIRS), acute respiratory distress syndrome (ARDS), and multiple organ dysfunction syndrome (MODS) [1–6]. For example, we recently reported that the mean base deficit and the mean alveolar–arterial gradient during the first 2 days after burn, as well as the age, burn size, and inhalation injury, were independent predictors of mortality in burn patients [7]. Because of this, some authors have sought alternate systemic or regional measures of resuscitation adequacy.

Of the various regional organ systems, the gastrointestinal tract is particularly vulnerable to ischemia during burn shock. In dogs with 40% burns, blood flow to the small intestine and kidney (but not to the stomach or colon) was decreased at 1 h after burn [8]. In sheep with 40% burns and inhalation injury

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0305-4179/$32.00
doi:10.1016/j.burns.2006.11.013

Report Documentation Page

Form Approved
OMB No. 0704-0188

1. REPORT DATE	2. REPORT TYPE	3. DATES COVERED
01 NOV 2007	**N/A**	**-**

4. TITLE AND SUBTITLE	5a. CONTRACT NUMBER
Intestinal and gastric tonometry during experimental burn shock	5b. GRANT NUMBER
	5c. PROGRAM ELEMENT NUMBER
6. AUTHOR(S)	5d. PROJECT NUMBER
Cancio L. C., Kuwa T., Matsui K., Drew G. A., Galvez Jr. E., Sandoval L. L., Jordan B. S.,	5e. TASK NUMBER
	5f. WORK UNIT NUMBER
7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)	8. PERFORMING ORGANIZATION REPORT NUMBER
United States Army Institute of Surgical Research, JBSA Fort Sam Houston, TX 78234	
9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)	10. SPONSOR/MONITOR'S ACRONYM(S)
	11. SPONSOR/MONITOR'S REPORT NUMBER(S)

12. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for public release, distribution unlimited

13. SUPPLEMENTARY NOTES

14. ABSTRACT

15. SUBJECT TERMS

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19a. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			
unclassified	**unclassified**	**unclassified**	**UU**	**6**	

resuscitated according to the Parkland formula, Sakurai et al. demonstrated a decrease in ileal blood flow to 64% of baseline by 6 h after burn. At the same time, cardiac index only decreased to 83% of baseline and renal blood flow, unlike Asch's study, remained unchanged [9]. Ramzy et al. in a rat model used laser Doppler flowmetry to demonstrate decreased jejunal blood flow at 2–4 h. postburn [10]. The effect of these changes in intestinal blood flow on outcome was suggested by Levoyer et al., who found that increased intestinal permeability, which may result from ischemia, was associated with increased infection risk in burn patients [11].

The vulnerability of the gastrointestinal tract to after burn ischemia after burn generated interest in the use of tonometry during burn shock. Venkatesh performed gastric tonometry and subcutaneous gas measurements in burn patients; the gastric intramucosal pH (phi) and the subcutaneous PO_2 decreased, and the subcutaneous PCO_2 increased, with burn shock [12]. Lorente et al. found that the gastric intramucosal-intraarterial PCO_2 gap during the first 12 h after burn was significantly different between survivors and nonsurvivors of severe burn. However, only age, burn size, and the oxygen delivery 6 h after burn independently predicted mortality [13]. Recently, Holm et al. reported no correlation between the pHi (or the PCO_2 gap) and systemic hemodynamic variables or in-hospital survival in burn patients. These authors concluded that gastric tonometry is unsuited for monitoring splachnic perfusion during burn shock [14]. Since tonometry is proposed as a more sensitive indicator of regional ischemia than are global indices, and since mortality may result from multiple factors in addition to resuscitation adequacy, whether tonometric information could be used to guide resuscitation remains undetermined.

In view of this continued controversy, the purpose of this study was to evaluate the utility of tonometry during burn resuscitation in a porcine model of severe thermal injury. We hypothesized that gastric and ileal pHi would deteriorate after burn, and that delayed fluid resuscitation would restore these values.

2. Methods

This study was approved by the institutional Animal Care and Use Committee. The care of all animals was in accordance with the guidelines set forth by the Animal Welfare Act and other federal statutes and regulations relating to animals and studies involving animals and by the 1996 *Guide for the Care and Use of Laboratory Animals* of the National Research Council. All animals were maintained in a facility approved by the Association for Assessment and Accreditation of Laboratory Animal Care International.

Eleven male Yorkshire swine (weight 23.3 ± 0.9 kg) were used for this study. After an overnight fast, they were sedated with intramuscular tiletamine/zolazepam (3 mg/kg, Ft. Dodge Laboratories, N.W. Fort Dodge, Iowa, USA) and atropine (0.1 mg/kg, Vedco Inc., St. Joseph, Missouri, USA). Ten minutes later, general anesthesia was induced using isoflurane (Ohmeda Caribe Inc., Gayama, Puerto Rico) administered through a nose cone. The pigs were then intubated with a 6.0–8.0 mm endotracheal tube (The Kendall Company, Mansfield, Massachusetts, USA). The left carotid artery was cannulated with a 1.9 mm catheter (Becton Dickinson and Company, Sparks, Maryland, USA) for blood-pressure monitoring. An external jugular vein was cannulated with a 7 F introducer (Arrow International Inc., Reading, Pennsylvania, USA), for infusion of drugs and fluids. A pulmonary artery thermodilution catheter was inserted through the jugular venous introducer. A celiotomy was performed. A Foley catheter was inserted via a cystostomy and secured with a purse-string suture. A carbon-dioxide-permeable air tonometer (Tonocap, Datex-Engstrom, Tewksbury, MA) was placed through an ileotomy into the terminal ileum, and was secured with a purse-string suture. A second tonometer was placed orogastrically.

Postoperatively, animals were transported to an animal intensive-care unit where they were nursed on foam pads and continuously monitored. They were continuously anesthetized with intravenous infusions of ketamine and propofol throughout the remainder of the 24-h study. The position of the animals was changed every 2 h. Enteral nutrition and H_2 blockers were not given. Animals were mechanically ventilated in the volume-control mode (Servo 900C, Siemens, Munich, Germany) throughout the experiment. Tidal volume was maintained at 13 ml/kg, the fraction of inspired oxygen was 0.4, and the respiratory rate was adjusted to maintain the partial pressure of carbon dioxide in arterial blood ($PaCO_2$) at approximately 30–40 mmHg.

After vital signs stabilized, baseline studies were done. A 70% full-thickness scald injury was then induced by immersion in water at 97 °C for 30 s. Anatomic landmarks were used to ensure uniformity in burn size among animals. Animals were then returned to the animal intensive-care unit. No fluids were provided until 6 hours after burn. Between hours 6 and 8, delayed resuscitation was performed according to the Parkland formula (4 ml of lactated Ringer's solution/% burn/kg body weight/24 h), with infusion of the volume predicted for the first 8 h over postburn hours 6–8. Between hours 8 and 24 after burn, the lactated Ringer's solution was titrated to achieve a urine output of 1–2 ml/kg/h. Twenty-four hours after injury, animals were euthanized with an overdose of pentobarbital (Fatal-Plus, Dearborn, MI).

Arterial blood gases (ABGs) and pulmonary arterial (PA) catheter data were measured every 6 h. Gastric and ileal regional PCO_2 ($PrCO_2$) were measured continuously by air tonometry and recorded hourly (Tonocap, Datex-Engstrom, Tewksbury, MA). The gastric and ileal pHi were calculated every 6 h by means of the modified Henderson–Hasselbach equation:

$$\mathrm{pHi} = 6.1 + \log_{10}([\mathrm{HCO_3^-}]/(0.03\,\mathrm{PrCO_2})).$$

The gastric and ileal PCO_2 gaps were calculated every 6 h according the formula:

$$\mathrm{PCO_2\ gap} = \mathrm{PrCO_2} - \mathrm{PaCO_2}.$$

The tonometric data were not used to alter the care of the animals.

Statistical analysis employed paired-samples *t*-tests comparing the baseline to hour 6 after burn, hour 6 to hour 24, and the baseline to hour 24 after burn (SPSS Version 14, Chicago, IL). *p* Values were Bonferroni-corrected for three non-orthogonal

Table 1

	Hour 0	Hour 6	Hour 12	Hour 18	Hour 24	*p* (0–6)	*p* (6–24)	*p* (0–24)
Heart rate	115 ± 5	176 ± 11	106 ± 4	111 ± 5	130 ± 6	**<0.001**	**0.018**	0.304
ABP mean	80 ± 3	71 ± 4	68 ± 4	64 ± 3	62 ± 4	0.056	0.344	0.059
CO	2.4 ± 0.4	1.5 ± 0.2	2.6 ± 0.6	2.6 ± 0.5	2.2 ± 0.2	**0.033**	0.056	0.996
PCWP	5 ± 0	3 ± 1	9 ± 1	10 ± 1	11 ± 2	**0.006**	**0.003**	**0.011**
pH Arterial	7.49 ± 0.02	7.44 ± 0.01	7.53 ± 0.01	7.49 ± 0.01	7.39 ± 0.05	**0.012**	0.567	0.211
Base excess	4.9 ± 0.8	1.0 ± 0.9	3.7 ± 0.7	2.9 ± 0.6	−1.0 ± 2.0	**<0.001**	0.987	0.041
HCO_3	28.2 ± 1.0	24.3 ± 0.9	26.6 ± 0.7	39.6 ± 13.5	23.9 ± 1.4	**<0.001**	0.999	0.062
$PaCO_2$	38.6 ± 2.6	36.6 ± 1.9	32.8 ± 1.3	35.2 ± 1.5	40.0 ± 2.7	0.593	0.449	0.955
$PrCO_2$ gastric	55.2 ± 4.7	71.2 ± 6.2	63.7 ± 7.1	63.8 ± 6.2	63.6 ± 4.3	0.079	0.336	0.703
$PrCO_2$ ileal	59.3 ± 3.4	85.4 ± 5.1	50.6 ± 2.3	53.2 ± 4.6	56.4 ± 4.1	**<0.001**	**0.003**	0.742
pHi gastric	7.34 ± 0.04	7.16 ± 0.04	7.27 ± 0.04	7.37 ± 0.10	7.18 ± 0.06	**0.006**	0.985	0.211
pHi ileal	7.31 ± 0.02	7.08 ± 0.03	7.35 ± 0.02	7.41 ± 0.09	7.22 ± 0.06	**<0.001**	0.209	0.617
PCO_2 gap gastric	19.2 ± 5.6	34.2 ± 7.1	30.9 ± 6.7	23.0 ± 4.3	25.0 ± 6.4	0.129	0.266	0.844
PCO_2 gap ileal	20.9 ± 3.0	48.7 ± 5.7	17.9 ± 2.2	18.0 ± 4.8	18.8 ± 4.3	**0.003**	**0.003**	0.891

ABP, arterial blood pressure. CO, cardiac output by thermodilution. PCWP, pulmonary capillary wedge pressure. HCO_3, arterial bicarbonate. $PaCO_2$, partial pressure of carbon dioxide in arterial blood. PrCO2, partial pressure of carbon dioxide, measured by tonometry. pHi, intramucosal pH, calculated from $PrCO_2$. PCO_2 gap, $PrCO_2 - PaCO_2$. *p* (0–6), *p* value for paired samples *t*-test, hour 0 vs. hour 6. *p* (6–24), *p* value for paired samples *t*-test, hour 6 vs. hour 24. *p* (0–24), *p* value for paired samples *t*-test, hour 0 vs. hour 24. Significant *p* values are indicated in bold.

comparisons. A *p* value of 0.05 was considered significant. Data are presented as means ± S.E.M.

3. Results

Results are given in the Table 1. Burn shock (i.e., at 6 h after burn) was associated with significant tachycardia, and decreases in the cardiac output, pulmonary capillary wedge pressure, arterial base excess, and calculated arterial bicarbonate level. A decrease in the mean arterial blood pressure was not significant. Aggressive resuscitation with crystalloid increased the pulmonary capillary wedge pressure above baseline, and restored the cardiac output to values not different from baseline, but failed to improve the base excess at 24 h. With respect to tonometric variables, the ileal variables uniformly changed with burn shock, and were restored to values not different from baseline by resuscitation. Similar changes in the gastric variables were statistically significant only for the pHi during burn shock. Consistent with this, hourly data (Figs. 1–5) indicate that ileal $PrCO_2$ responded more dramatically to burn shock and more convincingly to resuscitation than did gastric $PrCO_2$.

Fig. 1 – Heart rate as a function of time. Postburn hour is given on the abscissa in this and subsequent figures.

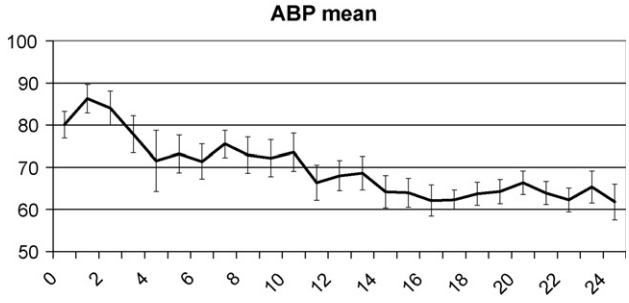

Fig. 2 – Mean arterial blood pressure, mmHg.

4. Discussion

The principle finding in this study was that ileal tonometry provided continuous data of potential utility for the resuscitation of animals with severe burn. Gastric tonometric data, by contrast, did not respond in as significant a fashion to burn shock and resuscitation.

The difference between the intestinal and gastric tonometric values may indicate the relative sensitivity of the small intestine to burn-shock-induced changes in regional perfusion [8,9]. Relatedly, in a porcine model of hemorrhagic shock, Walley et al. found that jejunal but not gastric tonometry correlated with superior mesenteric vein PCO_2 and oxygen delivery [15]. Puyana et al. measured small bowel and gastric pHi via implanted electrodes during a hemorrhagic shock study in swine. They reported more rapid and greater changes in small bowel pHi than in gastric pHi [16].

However, methodological problems with gastric tonometry have been identified and may have influenced the results of our study. Increased acid production may increase gastric CO_2 production, unless H_2 antagonists are used during resuscitation [17]. We did not do so in the present study. On the other hand, gastric feeding may also increase intraluminal CO_2 levels [18]; animals were not fed during our study. Saline

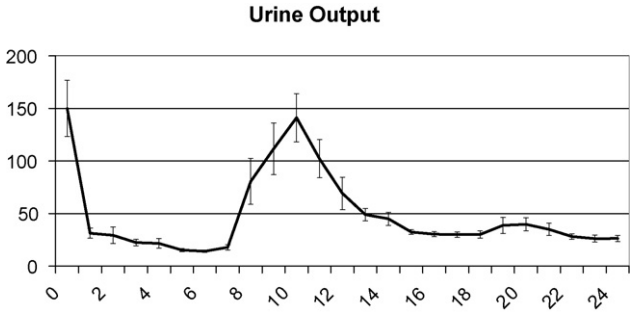

Fig. 3 – Urine output, ml/h.

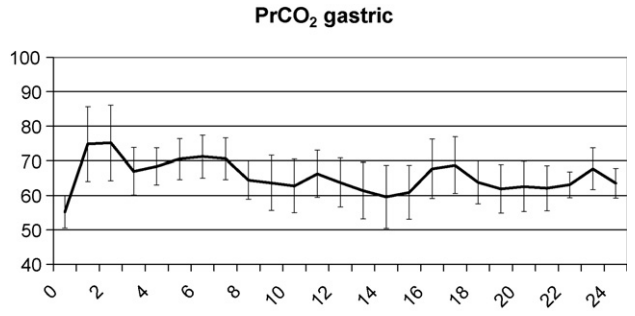

Fig. 4 – Gastric regional PCO_2, mmHg.

tonometers, which are difficult to use and require a dwell time to enable the CO_2 to diffuse into and dissolve in the saline, have been shown to yield erroneously low and poorly reproducible results [19]. We avoided this problem by using an air tonometer in our study.

The concept of intramucosal pH estimation by tonometry depends on the assumption that intramucosal HCO_3 and arterial HCO_3 are equilibrated. During rapidly changing physiologic states, this assumption may be incorrect. Thus, several authors have proposed using the PCO_2 gap instead of the pHi as a measure of intramucosal ischemia [20,21]. We observed similar trends in ileal pHi, $PrCO_2$, and PCO_2 gap in our study. However, the potential advantage of continuous $PrCO_2$ readings, which, unlike pHi and PCO_2 gap calculations are available from the air tonometer, does stand out from our study.

Considerable clinical experience has been amassed with gastric tonometry in non-burn critically ill patients [22–24]. This literature is controversial, but indicates that gastric

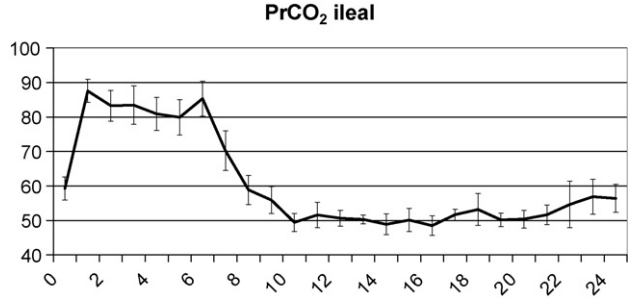

Fig. 5 – Ileal regional PCO_2, mmHg.

tonometry is one of the few end-organ-specific monitors approved for clinical use [23]. The Eastern Association for the Surgery of Trauma (EAST) recently published a clinical practice guideline on endpoints of resuscitation. They recognized that tonometry can provide data that can be used to predict risk of MODS or death [25]. How best to utilize the data from a tonometer has not been determined [24].

We did not use the tonometric data to guide fluid resuscitation in our study. We are not familiar with any studies in which this was done. Such a study would help place the utility of tonometry during burn resuscitation in perspective. However, as abdominal compartment syndrome is increasingly identified during burn resuscitation [26], tonometry may provide information about the adequacy of gastrointestinal perfusion in patients at risk for this syndrome. Thus, PCO_2 gap was predictive of abdominal compartment syndrome in trauma patients [27]. Furthermore, supranormal fluid resuscitation led to an increase in PCO_2 gap and in the risk of the syndrome [28]. We envision that during a difficult burn resuscitation, tonometry may help determine at what point a lot of fluid becomes too much fluid, and to gauge the risks and benefits of clinical interventions in that setting. On the other hand, Andel et al. used tonometry to demonstrate that early duodenal feeding was associated with a decrease in gastric PCO_2 gap, rather than a worsening of supply–demand mismatch in that organ [29]. Because burn patients are vulnerable to intestinal injury [11] or even infarction [30], tonometry may be useful to uncover occult ischemia during burn resuscitation.

Although we would like to recommend intestinal intubation and tonometry based on the results of this study [15], and despite the fact that jejunal feeding is the preferred practice at many centers, we recognize the technical difficulties involved in achieving this within the first hours of admission. Other authors have explored tonometry via other locations. The sublingual route is easier to access and does not require acid-suppressive therapy or discontinuation of enteral feeding. In several studies data obtained from this route has correlated well with that obtained from the stomach [31–33]. Rectal pH measurement and bladder tonometry [34,35] have been evaluated in animal models. The sigmoid route is particularly appealing for patients at risk for ischemia of that organ, such as during repair of abdominal aortic aneurysm [36]. Intraperitoneal tonometry can be performed [37].

In one hemorrhagic shock study, directly measured pHi reacted more quickly, and to a greater extent, than did pHi measured by tonometers [16]. In order to circumvent the shortcomings of tonometry, other methods of measuring or estimating intramucosal pH or metabolic status have been developed [38,39]. It seems likely that as these technologies improve, it will become desirable to monitor gut perfusion during shock more routinely, in order to provide what has been appropriately termed "Total Splachnic Resuscitation" [40].

In conclusion, in a porcine model of severe thermal injury and delayed resuscitation, we demonstrated the responsiveness of ileal tonometry to shock and resuscitation. Gastric tonometry responded in a similar fashion, but to a lesser extent. This study suggests that tonometric data may be useful during burn resuscitation, but leave open the question of how best to use the data.

Acknowledgement

Funded by the Combat Casualty Care research program, U.S. Medical Research and Materiel Command, Fort Detrick, Maryland, USA.

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